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EDITORIAL

THOSE of our readers who studied Dr. Gillespie's article on chloroform in the last issue of the journal will have noticed and possibly been astonished at some of the figures in the tables; for example there is a higher incidence of respiratory complications after chloroform than after any other anæsthetic. This Dr. Gillespie explains, is entirely due to the " Adverse pre-operative condition of the patients . . . . . more than a third of them suffered from advanced malignant disease ".

Again, with reference to respiratory complications following spinal anæsthesia, there are more than four times as many major post-operative complications after Nupercain as after Pontocain (amethocain). This appears to be due mainly to the fact that Nupercain is used for upper abdominal sections owing to its greater length of action, whilst Pontocain is used for lower abdominals such as appendicectomy. It seems unlikely that this explanation is adequate. Experience suggests that when a surgeon decides to open the abdomen, it is wise for the anæsthetist to secure analgesia to the nipple line.

Another point in Dr. Gillespie's paper is the use of the word " Tensity ". It will be of particular interest to those

who value nice distinctions. In what does it differ in meaning from " Tension ". The Murray dictionary gives the meaning of " Tensity " as " The quality or condition of being tense ". " Tension " is defined as the " Condition of being strained or stretched ". No clear distinction so far. References given in the dictionary show that " Tensity " is generally used in relation to physical structures, e.g. " That robust tensity of the fibres which makes strong people the less liable to accident ", but one reference from Carlyle's " Frederick the Great " is an exception. He speaks of " Such a tensity of spirit ". To-day the word " Tensity " is little used, but when it is, as in Dr. Gillespie's article, it refers to the condition of the muscles or other fibres, one of whose qualities is elasticity. Tension indeed has much the same meaning, but though we may and often do talk of the " Tension " in Europe, and use the word in connection with the emotions, the state of the feelings, we should scarcely speak of the " Tensity " in Europe. Anæsthetists will have no difficulty in making the distinction, for when the tensity of the abdominal muscles is raised the emotional tension in the atmosphere of the operating theatre is apt to be raised too.

At a meeting of the Anæsthetic section of the Medical Society of Manchester in August last, Dr. Courtin of the Mayo Clinic, whose paper we publish, gave a short account of the automatic regulation of the depth of anæsthesia by means of brain waves.

Anæsthesia modifies brain waves and by means of a very ingenious instrument their modifications are used to regulate the depth of anæsthesia. After the lecture, various speakers had " their say ", but it was astonishing to hear

that some of them thought that the automatic regulation of anæsthesia was something new. Anæsthetists are well aware that mortality from anæsthetics is roughly less than a tenth of that due to surgical interference. Yet anæsthetists are all the time dealing with the most important vital mechanisms while the surgeon is dealing with comparatively unimportant organs like the appendix, gall bladder or limbs. How comes it about that there is such discrepancy between the mortalities? Modesty forbids us to think we are ten times as clever as the surgeons and they doubtless would support that view. What then? Surely the patient's own R.C. corrects our excesses, takes over our deficiencies and generally smooths out the irregularities of even the youngest and most modern anæsthetist. Were it not for this steady friend, how horrible would be the massacre! Can it be that he has gone unrecognized and that there are anæsthetists who attribute the lives of their patients and their own prestige to their own skill?

It is of course true that the main body of anæsthetists are diffident, modest and shy, but there are some rash fellows who supersaturate their patients with N_2O, bring their respiration to nought with cyclopropane, or paralyse it with the deadly arrow poison from South America.

Do they not suppose that the co-ordinated activities of the vital mechanisms are of more value as the guardian of a man's life than any or many anæsthetists? Do they imagine that the comparatively coarse, clumsy contraptions of to-day can compare with the thousand-million-year-old mechanisms of our Maker, whose known niceties of adjustment, whose instant response to change, whose protective powers, are perpetually in action to preserve constant the conditions of life in the internal environment?

The ingenious instrument of which Dr. Courtin told us

is just a method of showing us that brain waves are in harmony with R.Cs. We might have guessed as much but it is satisfactory to have it confirmed.

As some of our readers know, Dr. Gray has been away in Denmark giving a course of lectures on anæsthesia in Copenhagen. As an example of the work done in that city, we are printing a short article on the use of pethidine in intubation of the larynx.

Overheard at a London Hospital:

First Anatomy Student: "We have got a good supply of bods this year".

Second Anatomy Student: "I wonder where they come from? I suppose they are the people who die under anæsthetics".

It gives us the greatest pleasure to congratulate Dr. Minnitt on his recently acquired honours. The Henry Hill Hickman Medal is surely a most appropriate reward for one who has so extended the use of nitrous oxide. The honorary fellowship of the R.C.O.G. is no less so, for one who has done so much to relieve the pain that flesh is heir to.

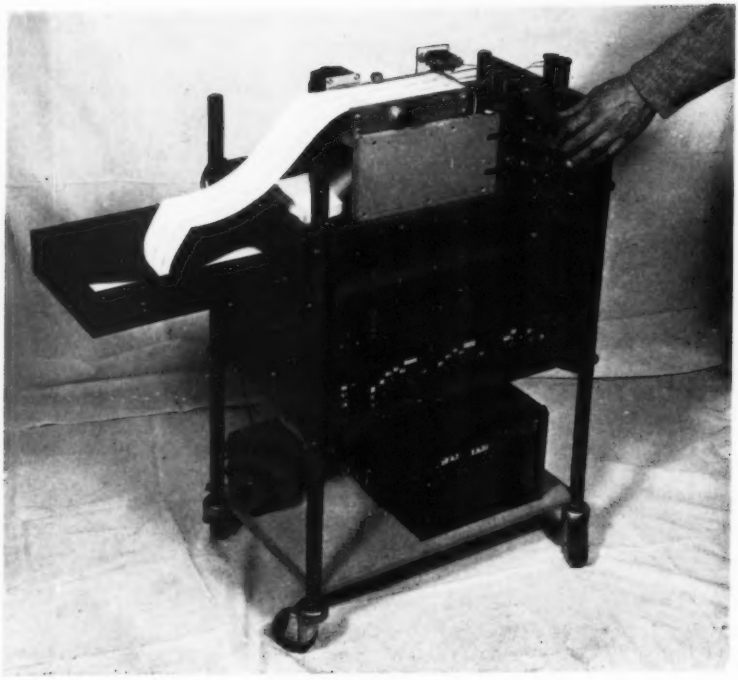

FIG. 1

Apparatus used in recording electro-encephalographic and electro-cardiographic patterns during surgical operations

THE VALUE OF CONTINUOUS ELECTRO-ENCEPHALOGRAPHIC AND ELECTRO-CARDIOGRAPHIC TRACINGS TO THE ANÆSTHETIST DURING SURGERY

By RAYMOND F. COURTIN

THE investigation into the effects of nitrous oxide-ether anæsthesia on the electro-encephalogram by Courtin, Bickford, and Faulconer (1950) showed that a constant relationship existed between the changes in the electro-encephalographic pattern and the depth of anæsthesia. The apparatus (Fig. 1) used for most of the investigation consisted of a two-channel inkwriting crystograph designed for compactness and high discrimination against interference. One channel was used for recording the electro-encephalogram, the other the electro-cardiogram. Electro-encephalographic electrodes were solder disks secured by collodion and filled with electrode jelly through a central hole. The frontal electrode was placed on the frontal eminence about 2.5 cm. from the midline, and the occipital electrode was directly posterior to the frontal electrode, about 4 cm. from the occipital protuberance. A grounded lead was attached to the mastoid process.

The changes in the electro-encephalographic pattern were classified into seven levels (Fig. 2). The first and second levels occurred during induction. The first level (flat) is characterized by an almost flat tracing, in some cases showing fast frequencies of 20 to 30 cycles per second, and lasting in a normal induction for about seven minutes. The second level (rhythmical) consists of high amplitude

waves, 200 to 300 microvolts, with a consistent frequency of 2.8 per second; these waves usually appeared suddenly but seldom lasted for more than a minute. The third, fourth and fifth levels were found to be suitable for all surgical procedures, depending on the degree of muscular relaxation required. The third level (complex) has a complex appearance but the waves are still of high amplitude and there is continuous activity. During the fourth level (slight suppression) short periods (one to three seconds) of

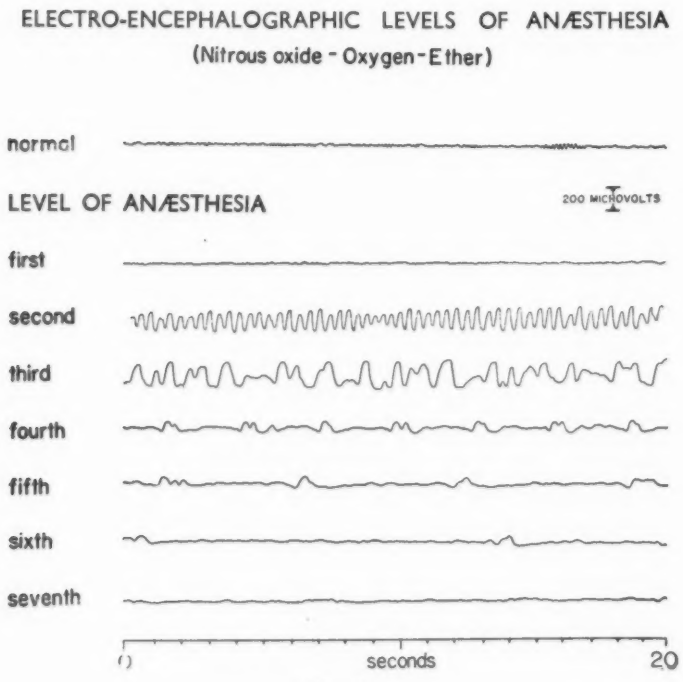

FIG. 2

Electro-encephalographic patterns characteristic of successive electro-encephalographic levels of anæsthesia

relative cortical inactivity appear and the amplitude of the waves is less than in the preceding level. The fifth level (moderate suppression) differs from the fourth in the duration of the period of cortical inactivity, lasting here between three and ten seconds. The amplitude of the waves is again less than in the preceding level. The sixth (severe suppression) and seventh levels (complete suppression) were considered to indicate unnecessarily deep anæsthesia and were characterized by cessation of cortical activity, the latter showing complete absence of waves at the gain employed.

During the investigation, records were obtained which were of particular interest; records in two cases are described in detail.

Case 1

A white woman aged 60 years underwent operation for cholecystectomy. Premedication consisted of 1½ grains (100 mg.) of pentobarbital sodium (Nembutal) one and a half hours before operation, 1/6 grain (10 mg.) of morphine sulphate and 1/150 grain (0.43 mg.) of atropine sulphate one half hour before operation. Induction was performed with nitrous oxide and oxygen, 80:20 mixture, followed by ether. In addition to recording the electro-encephalographic patterns, a mobile photographic recording oscillograph was used as described by Faulconer and Ridley (1950). This oscillograph recorded the following physiologic variables: (1) depth and rate of respiration; (2) arterial oxygen saturation; (3) analysis of the inspired gases; (4) instantaneous heart rate and (5) electro-cardiograph.

The further description of this case is divided into (1) clinical observations, (2) electro-encephalographic record and (3) subsequent data obtained from the photographic record.

Clinical Observations. Clinically, the patient's induction was normal. At seven and a half minutes from the start of the anæsthesia a Guedel airway was inserted; the ether concentration was increased rapidly during the first ten minutes and the nitrous oxide–oxygen ratio was reduced to 60:40 at nine minutes. At this time the patient's breathing was fast and had increased in volume. At twelve minutes, she suddenly became apnœic, which

led to reduction of the ether concentration and increase of the oxygen flow. Because the patient did not start to breathe spontaneously, an intratracheal tube was introduced orally and artificial respiration by manual bag pressure was instituted during two periods of half a minute at thirteen and one half and fifteen minutes. At seventeen minutes, the patient resumed spontaneous breathing, the peritoneum was opened at nineteen minutes and the subsequent course of the anæsthesia was uneventful.

Electro-encephalographic Record (*Fig.* 3). The pattern characterizing the second level appeared at five minutes after the start of the anæsthesia, the third level at six and one fourth minutes, the fourth level at eight and one fourth minutes and at ten minutes appeared an atypical pattern which has been observed

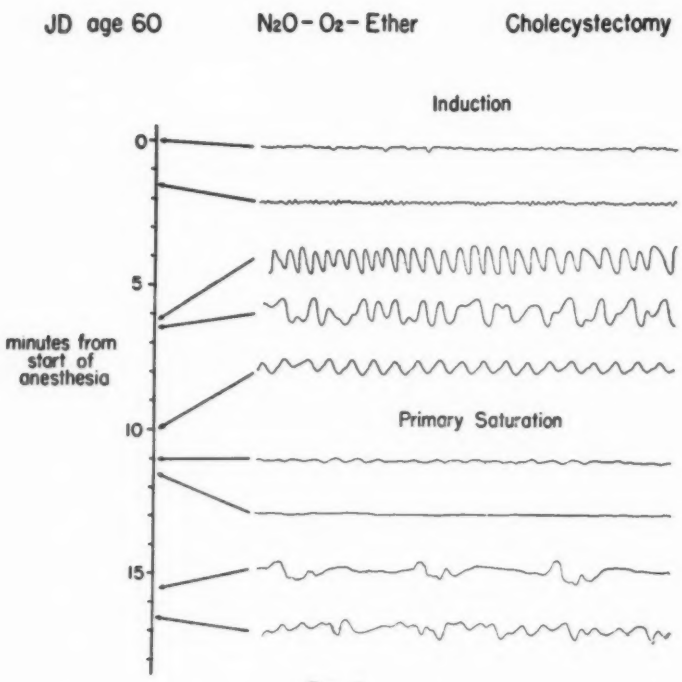

FIG. 3

Portions of the electro-encephalographic tracing of case 1

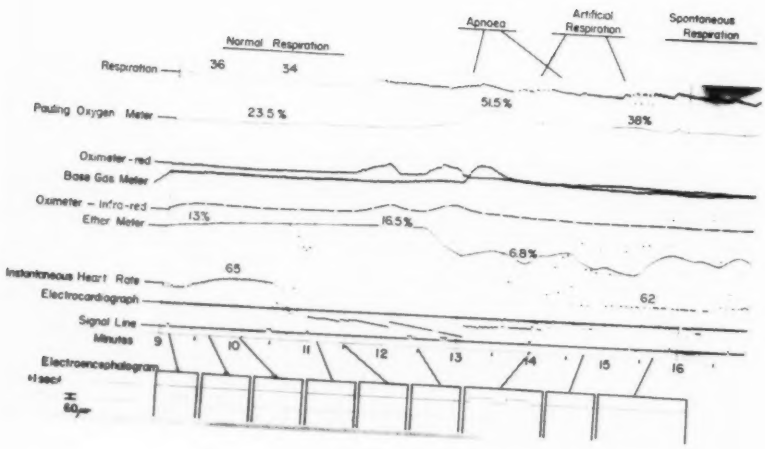

FIG. 4

Portion of the actual photographic record of the physiologic variables occurring between nine and seventeen minutes in case 1

on only a few occasions. This atypical pattern was followed by the seventh level at ten and one half minutes. This case occurred early in the investigation. Complete absence of cortical potentials having not previously been observed, the immediate impression was that the recording apparatus had failed! From later study of the photographic record and subsequent experience it was realized that this complete absence of cortical potentials indicated dangerously deep anæsthesia. The interval of one and one fourth minutes between the appearance of the seventh level at eleven minutes and the action of the anæsthetist to reduce the ether concentration at twelve and one fourth minutes should be noted, as this suggested that the electro-encephalographic tracing could give warning of untoward complications. The seventh level continued until fifteen and one half minutes when rapid return of cortical activity occurred through the fifth and fourth level to the third level at seventeen minutes.

Subsequent Data obtained from the Photographic Record. Data from nine to seventeen minutes are shown in Figure 4. The

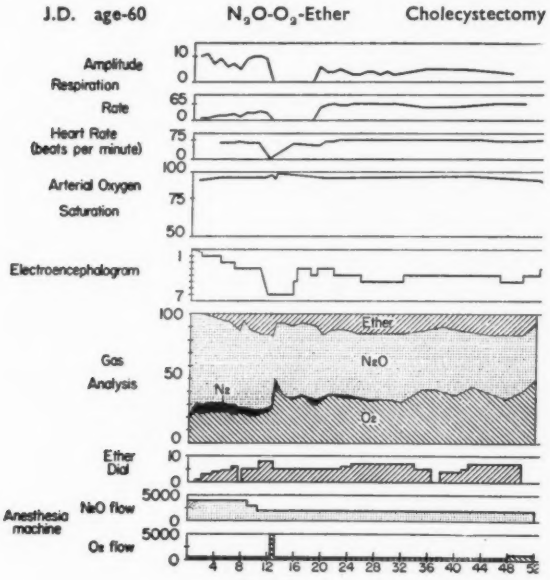

FIG. 5

Graphic analysis of the photographic record of case 1

hyperpnœa until just before twelve minutes, followed by the period of apnœa and the artificial respiration, is clearly visible on the record. At the time of the complete absence of the cortical potentials, it will be seen that the ether concentration in the inspired air had risen to 16.5 per cent. The effect of the anæsthetist's reducing the ether concentration and increasing the oxygen flow at twelve and one fourth minutes is seen in the ether meter and Pauling oxygen meter tracings. The instantaneous heart rate record is interesting; between ten and eleven minutes a rate of 65 per minute was recorded. Unfortunately, on this reproduction of the record it is impossible to read the electrocardiographic tracing because of the slow speed of recording, but on careful scrutiny of the original record, no QRS complexes are visible between eleven and one half and twelve minutes and again between twelve and one half and thirteen minutes. The period of apnœa had no marked effect on the arterial oxygen saturation, only a slight decrease occurring at twelve minutes. The simultaneous rise in both the red and infrared tracings indicates a decrease in blood pressure which, it will be seen, occurred at the same time as the absence of QRS complexes in the electro-cardiographic tracing. A complete analysis of the photographic record is shown in figure 5.

Case 2

A white man aged 55 years, height 72 inches, weight 196 pounds, underwent operation for a skin graft of the left thigh. Three days previously this patient had been scheduled for the same operation but the operation had to be postponed. On the latter occasion premedication consisted of 1½ grains (100 mg.) of pentobarbital sodium (Nembutal) one and one half hours before operation, 1/6 grain (10 mg.) of morphine sulphate and 1/150 grain (0.43 mg.) of atropine sulphate one half hour before operation. Anæsthesia was induced with 2.5 per cent thiopentone sodium and after 16 cc. (400 mg.) had been injected intravenously, the patient's pulse became irregular and his blood pressure dropped from a preoperative level of 110/60 mm. of mercury to 60/40. His previous history was negative except for a burn in childhood for which he needed the skin graft; he was a complete teetotaler. It was decided as a result of the experience with the thiopentone sodium to use nitrous oxide, oxygen and ether. The premedication was 1/150 grain (0.43 mg.) of atropine sulphate. The induction was smooth but similar to the first record described previously in that the electro-encephalographic tracing showed the sixth level (severe suppression) early in the anæsthesia at

fifteen and one half minutes following the fourth level at fifteen minutes. It will be seen from figure 6 that during both the fourth and the sixth level, auricular premature contractions occurred with a marked increase to 124 in pulse rate from a preoperative rate of 66. This tachycardia and arrhythmia were accompanied by a considerable drop in blood pressure. The ether concentration was immediately decreased. The patient returned to the third level at nineteen minutes and the heart resumed a normal sinus rhythm. Later it was noticed that if the ether concentration was increased and consequently the electro-encephalographic pattern changed to the fourth level, signs of cardiovascular instability returned.

Three weeks later this patient again underwent operation for a similar procedure, and as a result of careful observation of the electro-encephalographic record anæsthesia was never allowed to become deeper than the third level; the anæsthesia was uneventful and no tachycardia, arrhythmia or drop of blood pressure occurred. For this patient, the ether concentration in the inspired air had to be kept considerably lower than that usually administered to a normal subject even in light ether anæsthesia. These were the first occasions on which recording the electro-encephalographic tracing was used clinically and not experimentally, and it was noted how easily a patient could be maintained in light ether anæsthesia by using this measure of electrical cortical activity.

COMMENT

Data on these cases have been presented because it is thought that continuous electro-encephalographic and electrocardiographic recording in the operating room may provide the anæsthetist with an additional aid to the administration and safe conduct of the anæsthetic.

In case 1 the patient's induction was rapid and the accompanying hyperpnœa probably resulted in a dangerous concentration of ether in the cerebral circulation. Appreciation of the electro-encephalographic changes would have prevented the ensuing five minutes of apnœa and severe cardiovascular disturbance.

In the second case the patient had an idiosyncrasy to anæsthetic agents which showed itself as a cardiac conduc-

tion defect. The electro-encephalogram provided a simple way of maintaining this patient in light anæsthesia which fortunately was all that was required for the surgical procedure. However, had it been necessary to provide muscular relaxation, this would have been safely obtained only by using one of the muscular relaxant drugs, an interesting point for those who still maintain that these drugs have no place in anæsthesia!

A series of cases has been started in which it is proposed to maintain light ether anæsthesia in level three and produce the required muscular relaxation with d-tubocurarine. Thus, using the electro-encephalographic pattern as a means of maintaining light anæsthesia, the objectives of this investigation will be:

1. To ascertain if the electrocardiographic changes associated with ether anæsthesia described by Lennox and his co-workers (1922) and Kurtz and his co-workers (1936) can be eliminated at this relatively light level of anæsthesia.

2. To ascertain if undesirable fluctuation or falls in blood pressure associated with deep ether anæsthesia (Goodman and Gilman, 1941) can be prevented.

3. To observe whether postoperative complications such as nausea, vomiting and hypotension can be reduced with this technique.

SUMMARY

Data on 2 cases have been presented to illustrate the value of continuous electro-encephalographic and electrocardiographic tracings to the anæsthetist in the operating room. There is a preliminary report of a study using light nitrous oxide ether anæsthesia and d-tubocurarine.

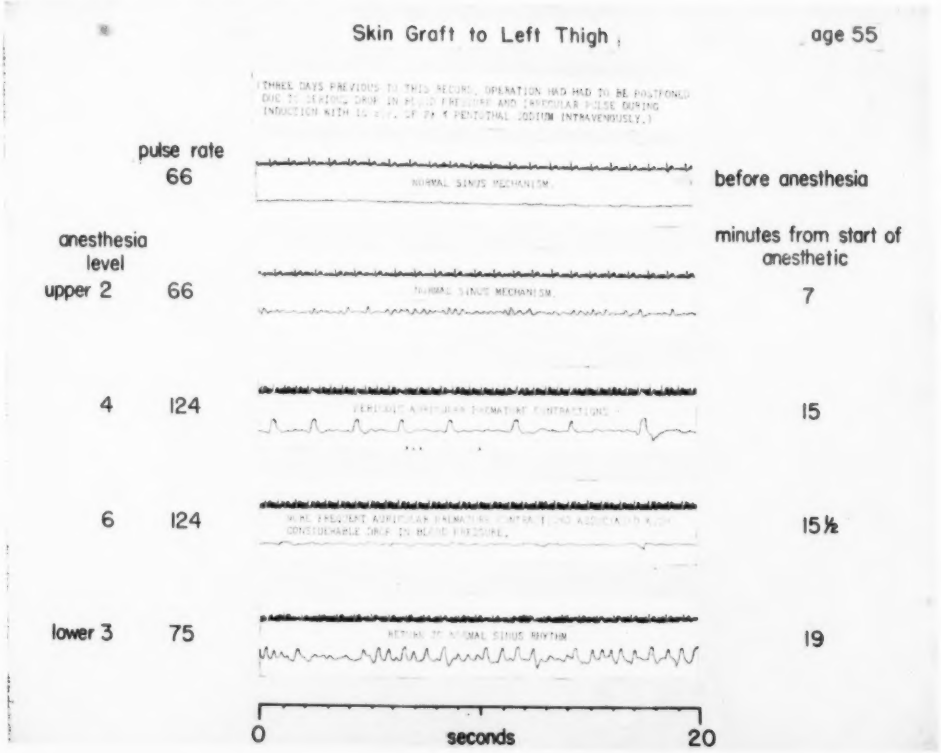

FIG. 6

Portions of the electro-encephalographic and electro-cardiographic tracings in case 2.

REFERENCES

1. Courtin, R. F., Bickford, R. G., and Faulconer, Albert, Jr. (1950), *Proc. Stff. Met., Mayo Clin.*, **25**, 197.

2. Faulconer, Albert, Jr., and Ridley, R. W. (1950), *Anesthesiology*, **11**, 265.

3. Goodman, L. S., and Gilman, Alfred (1941), "The Pharmacological Basis of Therapeutics; a Textbook of Pharmacology, Toxicology and Therapeutics for Physicians and Medical Students". The Macmillan Company, New York, p. 60.

4. Kurtz, C. M., Bennett, J. H., and Shapiro, H. H. (1936), *J. Amer. med. Ass.*, **106**, 434.

5. Lennox, W. G., Graves, R. C., and Levine, S. A. (1922), *Arch. int. Med.*, **30**, 57.

SPINAL ANÆSTHESIA

By E. FALKNER HILL

IN these days when curare has been introduced into clinical medicine in the extremely convenient form of an intravenous injection, when it produces such excellent relaxation of the abdominal musculature, and can be repeated as often as required and accompanied by any anæsthetic that the anæsthetist may fancy, it may well be that spinal analgesia will fall into disuse. Marcel J. Dallemagne (1949) in his book, *Aspects-actuels de l'Anesthésiologie*, says of spinal anæsthesia: "Elle ne presente ni plus ni moins de dangers que n'importe quel mode d'anesthésie, mais elle réclame de la part de celui qui la pratique, encore plus de décision et d'intelligence que toute autre technique."

It would be perhaps unreasonable to expect that anæsthetists generally should bother about acquiring that "plus de décision et d'intelligence" which spinal anæsthesia demands when they have at hand so simple a technique as curare requires. There will always be, however, a few who "scorn delights and live laborious days" and take the trouble to acquire a technique and an experience that will give them not only a fresh interest, but results at least as good as those of other methods.

Statistics published in the *British Journal of Anæsthesia*, January 1948, show a mortality of 0.045 for spinal anæsthesia and nearly double that for general anæsthesia. As the figures relate to 22,500 and 77,000 cases respectively, and as fully two-thirds of the anæsthetics were administered by junior members of the resident medical staff, they may be taken as relatively fair. The technique taught, and

for the most part used, originated from clinical experience and the experimental work, a description of which immediately follows.

A quarter of a century ago there were a few men who had given hundreds or even thousands of spinal anæsthetics. Babcock and Labat in America, Sebrechts in Belgium, Jonesco in Bucharest, Forgue and Basset, Le Filliatre and Delmas in France, Barker in England, are just a few of the names that come to mind.

Amongst these and other experts there was a general agreement that if the anæsthetic went too high, dangerous or even fatal results followed, and though there was complete unanimity on this matter, there the unanimity ended. The question of exactly how the dangerous situation was brought about was much discussed; the following explanations were offered:

(1) *Paralysis of respiration.* Koster and Kasman (1929), however, deny this. They say: " One of us has demonstrated the possibility of pouring a solution of 100 mg. (0.1 g.) of procaine hydrochloride in 4 c.c. normal saline directly on to the medulla and upper cervical cord of animals, with the production of complete anæsthesia and sleep, with no gross interference with respiration, as shown by a hook sewn into the diaphragm or into the intercostal muscles, the other end of which is attached to a lever recording on a moving drum." They explain this result as being due to the fact that in this strength ($2\frac{1}{2}$ per cent) conductivity of the posterior roots is interrupted but not the excitability of the respiratory centre or the cells of the phrenic nuclei or the fibres proceeding from those cells.

In 1930 Koster says: " Very little is known about the various mechanisms by means of which death is produced." Henderson and Johnston (1932) of Toronto, on the con-

trary, hold that procaine or other spinal anæsthetic has no effect on the respiratory centre but paralyses the phrenics. They say: " High concentrations of Novocain in the fourth ventricle do not produce death. The major danger of acute death arises from a depression of the phrenic roots."

(2) *Fall of blood-pressure.* Many writers have stressed a fall of blood-pressure as the cause of death under spinal anæsthesia, and the anæsthetic as the cause of the fall of blood-pressure. Maxson (1938) summarizes these views as follows: " Lowering of the blood-pressure to a point where the heart fails to get the needed nutrition for its own musculature, becomes unable to do its work and ceases to beat "; on the other hand Labat (1928) thinks that a very low blood-pressure is nothing " to worry about ". He says: " It has been repeatedly stated that some patients become pulseless on the operating table and that the blood-pressure is so low that no reading can be taken. This is quite true and should not cause worry."

(3) *By heart failure.* Forgue and Basset (1930) say: " Cependant, il est hors de doute que maintes fois, dans certaines conditions (hyperdosage, hyperpression de l'injection lombaire, hypersonstraction liquidienne) il faut admettre que ces phénomènes hypotensifs sont produit par la hauteur de l'impregnation toxique qui atteint les racines des nerfs cervicaux (affaiblissement de la respiration diaphragmatique) et peut arriver aux centres bulbaires c'est bien alors de moteur cardiaque qui est frappé; et le succès des injections adrénalinées intra-cardiac dans les syncopes rachianesthétiques, semble le prouver."

It would seem, then, that Forgue and Basset think there is a species of heart failure distinct from that following, and due to, primary respiratory failure.

(4) *By absorption of the anæsthetic into the blood-stream.* Thus Howard Jones, at the Eastbourne meeting of the B.M.A. in 1931, said: "The danger is from blood-absorption and paralysis of the vasomotor centre leading in some cases with excessive doses to severe collapse to a pulseless state."

(5) *Cerebral anæmia.* Labat (1928) says: "Any technique which rests upon the fundamental principle of circulatory disturbances and attempts to cushion by posture the abruptness of such disturbances is of the highest value."

THE EXPERIMENTS

The object of the experiments on cats about to be described was to determine the exact mechanism of death.

As it is unanimously agreed that when the anæsthetic goes "too high" we are in the presence of danger, if not disaster, the first experiment was obviously to put the anæsthetic "too high"; accordingly we put 0.5 ml. saline containing 10 mg. of Novocain into the cisterna magna, and Fig. 1 shows what happened. The points to notice are the following:

(1) an immediate marked rise of blood-pressure accompanied by a slowing of respiration;
(2) the slowing of respiration passes on into paralysis;
(3) this paralysis of respiration is followed by a would-be fatal fall of blood-pressure;
(4) artificial respiration arrests this fall of blood-pressure; and
(5) raises the blood-pressure to a height considerably above its level prior to the injection of Novocain.

We may conclude then that the respiration was paralysed, that the fall of blood-pressure was the direct result

of heart failure due to anoxæmia caused by the respiratory failure; also that the vasomotor centre was active, as evidenced by the extensive rise of blood-pressure to a height considerably higher than that prior to the injection.

As this fall of blood-pressure was the direct result of respiratory failure, we supposed that if we artificially respired the cat (Fig. 2), before we injected the Novocain, there would be no fall of blood-pressure. We were mistaken; the blood-pressure fell distinctly. How was the difference between these two tracings to be explained? In tracing (1) the blood-pressure had risen on applying artificial respiration, but in tracing (2) the blood-pressure had fallen in spite of the fact that artificial respiration had been maintained throughout. We realized that in the first experiment, owing to the respiratory paralysis, the blood had become charged with CO_2, and it was probable that this excess of CO_2 in the blood stimulated the vasomotor centre and so caused the rise of blood-pressure. In the second experiment (Fig. 2) there was no accumulation of CO_2 and so no rise of blood-pressure, and in all probability the artificial respiration over-oxygenated the blood and washed out the CO_2, hence the fall of blood-pressure in this experiment.

The next experiment (Fig. 3) in which we artificially respired the cat with air and 5 per cent CO_2 prior to the injection, seems to confirm the suggested explanations.

In the fourth experiment (Fig. 4) 10 mg. of Novocain in 1 ml. saline were injected into the lumbar region. There follows a paralysis of respiration and subsequent fall of blood-pressure an in experiment 1, but on artificial respiration the blood-pressure rises only to some 70 mm. Hg, as against approximately twice that much in experiment 1. It would seem that this injection has paralysed the vasoconstrictor nerves which emerge from the cord by means of the

white rami from the second dorsal to the second lumbar nerve. These nerves were not affected by the intracisternal injection of experiment 1, and hence when artificial respiration restored the animal they were strongly stimulated and caused the marked rise of blood-pressure.

In the fifth experiment (Fig. 5) we injected 0.1 ml. saline in which 10 mg. of Novocain was dissolved into the region of the phrenic nucleus, between the second and third cervical vertebræ. There followed a paralysis of the diaphragm, but the cat was able to keep itself alive by its intercostal muscles, which compensated for the failure of the diaphragm by a marked increase in activity.

The next question to decide was that of centre or phrenic paralysis or both. Koster and Kasman (1929) said neither were paralysed by a $2\frac{1}{2}$ per cent solution of Novocain. Henderson and Johnston (1932) deny paralysis of the centre but are of opinion that the phrenics are paralysed when the anæsthetic goes " too high ".

We have just seen in the previous experiment (Fig. 5) that the phrenics were affected and the diaphragm paralysed and that in spite of this the cat was able to support itself by its intercostals. If to such a cat, that can support life with its intercostals, we give an intracisternal injection of Novocain, 5 or 10 mg., one of two things will happen. If there is no such thing as a central paralysis, the intercostals certainly, and the diaphragm probably (the bulk of the injection being only 0.1 ml.), will both be in action. If on the contrary there is paralysis of the respiratory centre, then both diaphragm and intercostals will fail simultaneously and that is as shown in Figs. 9 and 10.

This constitutes the proof of the possibility of paralysing the respiratory centre. An inspection of Figs. 5 and 8 will show the marked difference between a phrenic paralysis

and a central paralysis. In Fig. 5 the contractions of the diaphragm occur at regular intervals, but each is slightly less than the preceding one until complete paralysis sets in. Meantime the intercostals increase their activity in order to compensate for the paralysis of the diaphragm. It seems as if the centre, entirely free from the anæsthetic, sends out its impulses regularly, but the more the anæsthetic influences the phrenics the less the response.

Fig. 8, on the other hand, shows quite a different picture. Each contraction is as great as the preceding one until paralysis sets in, but the interval between two adjacent contractions gradually increases. It seems as though the centre found it more and more difficult to send out its impulse, but when it did, the diaphragm always made the maximum response.

These two tracings are respectively characteristic of a phrenic and a central paralysis. It seems obvious that two such different pictures must be produced by different mechanisms.

What of the fall of blood-pressure as a cause of a death on the table? Fig. 11 shows a fall of blood-pressure to 60 mm. Hg as the result of 50 mg. of Novocain, equivalent to at least 1.5 g. in an adult man. Two further doses of the same amount failed to bring the blood-pressure lower. It is unlikely that any spinal anæsthetic, provided it does not affect the phrenics or respiratory centre, will lower the blood-pressure of an animal that is healthy below about 60 mm. Hg, and therefore will not cause death by itself. The blood-pressure of a healthy cat having been reduced to 60 mm. Hg by Novocain can be lowered still further by opening the abdomen and pulling on the stomach as seen in Fig. 12. This resembles the fall of blood-pressure often seen in the operating theatre.

FALL OF BLOOD-PRESSURE FROM THE CLINICAL ASPECT

Different writers give differing explanations for the fall of blood-pressure which they are accustomed to see after the injection of a spinal anæsthetic. Thus Pannett (1933) attributes it to a paralysis of the vasoconstrictors supplying the vessels going to the muscles paralysed by the anæsthetic; Forgue and Basset (1930) and Sebrechts (1930) think it due to the paralysis of the vasoconstrictors supplying the splanchnic area; Seevers and Waters (1932) to failure of inspiration to suck the blood into the heart, due presumably to paralysis of the lower intercostals; and Forgue and Basset (1930) suggest that in some cases it is due to " Le moteur cardiaque qui est frappé ".

It seems to be taken for granted that a fall of blood-pressure necessarily occurs after the injection of a spinal anæsthetic. This is by no means so. Whether the blood-pressure falls or not depends on the patient, what he is suffering from, the preparation of the patient, the use or not of ephedrine beforehand, as well as on the nature of the anæsthetic, its dose and whether it is heavier or lighter than the C.S.F. Thus a healthy robust man of not more than middle age, who has had a preliminary dose of morphia and atropine and 100 mg. of ephedrine, undergoing a gastro-enterostomy, will as a rule have no fall of blood-pressure if given 25 mg. of amethocain in a 4 ml. mixture of C.S.F. and the solution in which the amethocain is dissolved. This solution is heavier than C.S.F. and will give anæsthesia up to the nipple line. On the other hand, if the patient is fat, flabby or feeble; if he has been overdosed with omnopon and scopolamine; if the ephedrine has been omitted; then under these conditions when the vital mechanisms are below par the blood-pressure may well fall. Again the

nature of the disease is a determining factor. If the patient has very severe abdominal pain, the spinal anæsthetic relieves this with dramatic suddenness and the blood-pressure comes down with a run; this is an indication of the efficiency of the anæsthetic and a matter for rejoicing rather than worry (Fig. 21). The nature of the anæsthetic used markedly affects the blood-pressure. It is common experience that Stovain is much more likely to cause a fall of blood-pressure than procain. Nupercain especially in light solution, according to Sebrechts (1934), attacks the anterior roots of the spinal nerves more particularly than the posterior roots and so is liable to cause a fall of blood-pressure. Light solutions are more apt to cause falls of blood-pressure than heavy ones for the obvious reason that the brunt of their action will fall on the anterior roots rather than on the posterior. My own experience with light and heavy procain undoubtedly confirms this view. Whether the failure of inspiration to suck blood into the heart as stated by Seevers and Waters (1932) is a cause of a fall of blood-pressure or not I have no knowledge, but it is on all fours with Sebrechts's (1934) observation that light Nupercain anæsthesia is characterized by a slight degree of cyanosis. If in the course of an abdominal section the blood-pressure falls as it quite often does, it is *after* the abdomen is opened, and is due to traction on viscera supplied by the vagus and is apparently due to vagus inhibition of the heart. I am inclined to think that this can be avoided in most cases if a small dose of thiopentone be given before the exploration takes place; I have very frequently observed this and if the observation is confirmed by other anæsthetists with extensive experience of spinal anæsthesia, it would seem to suggest that the fall is in part due to the patient's anxiety roused by traction on the vagus.

As an illustration, I may mention the case of a doctor who was undergoing a gastro-enterostomy. He said to me afterwards: "When Mr. X put his hand into my abdomen I thought I was passing out, but you said to me 'Now, Doctor, this is a little uncomfortable but you are all right; the blood-pressure is A1.' I didn't seem to mind it." Fig. 17 shows an unusually well marked fall due to the exploration of the abdomen. Fig. 18 shows the blood-pressure record of a man aged 45. The surgeon asked for as long an anæsthesia as possible. It was in the days when I was using procain, and I gave the patient 300 mg. of procain. The operation was much shorter than expected, lasting only three quarters of an hour. The anæsthesia was up to the area supplied by the fourth cervical, as he could not feel towel clips inserted into the skin over the shoulder joint. He could, however, move his arms, could breathe freely, and had no fall of blood-presure. It is difficult to suppose that anterior roots higher than the fourth dorsal were in any way affected. Here, then, we see a difference of eight segments between anterior and posterior roots affected. Surely a demonstration of the greater safety of a solution heavier than C.S.F.

Fig. 19 shows the record of the blood-pressure of a patient about to undergo an abdominal section. The surgeon came half an hour later than expected. Here we see again that following the ephedrine and 260 mg. of procain the blood-pressure rose from 125 to 160 mm. Hg. Only after the peritoneum was opened did it fall below its height prior to the operation.

A surgeon in the course of an operation on the gall bladder sometimes asks for the bridge to be raised. In these circumstances, if the patient is a fat woman, as often happens, and if the bridge be sufficiently powerful, the

blood-pressure will fall, the heart will be weakened, the pulse will fail, and if the bridge is not put down at once the patient will die. Presumably the inferior vena cava is narrowed and the return of the blood to the heart obstructed.

Another type of case in which the spinal anæsthetic causes a fall of blood-pressure is that associated with a high blood-urea. Not all cases in which we find a high blood-urea are unsuitable for spinal anæsthesia; for example, a high blood-urea is frequently found in cases of intestinal obstruction and there is no anæsthetic so suitable as a spinal for these cases. On the other hand, there is a type of case associated with high blood-urea which, if given a spinal anæsthetic, dies in a few minutes unless the means of rescue are unusually efficient, and even so the issue is doubtful. I think it is this type of case referred to by Forgue and Basset (1930) when they say: "Il y a une catégorie de malades, chez lesquels nous avons appris à redouter la rachianalgésie et le choc quel provoque: ce sont certains hyperazotémiques, chez lesquels un trouble de l'équilibre des chlorures est associée à une rétention uréique marquée."

I have seen two such cases certainly, probably four. It will be sufficient to describe one of them. A man aged between 60 and 70 was diagnosed as intestinal obstruction owing to persistent vomiting. I was asked to give this patient a spinal anæsthetic. Two or three minutes after the injection the patient had a good pulse, breathing comfortably, able to answer questions. A minute later he was unconscious, the radial pulse had disappeared, and the breathing was scarcely perceptible. A tube was introduced into his trachea, oxygen blown in, artificial respiration from time to time, two or three doses of Coramine injected, and after $2\frac{1}{2}$ hours the patient was able to breathe without help.

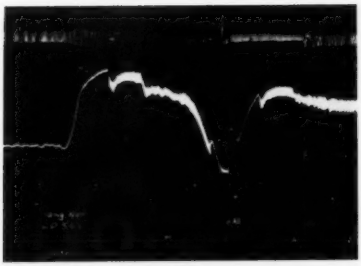

FIG. 1

Upper tracing, respiration. Lower tracing, blood-pressure. Notice the marked rise of blood-pressure immediately following the intracisternal injection of Novocain, the slowing and subsequent paralysis of respiration, and the ensuing would-be fatal fall of blood-pressure. Artificial respiration not only restores the blood-pressure to its former height but raises it considerably above that level.

FIG. 2

Notice the considerable fall of blood-pressure even though artificial respiration was in action (between the arrows) following the intracisternal injection of 10 mg. of Novocain.

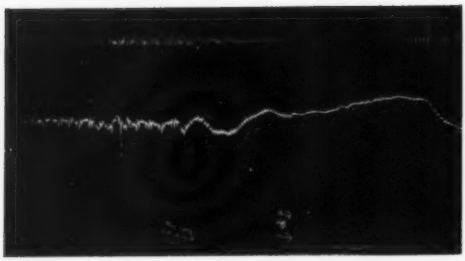

FIG. 3

Notice that there is no fall of blood-pressure following the intracisternal injection of 10 mg. of Novocain. The animal being artificially respired with 5 per cent CO_2 and air.

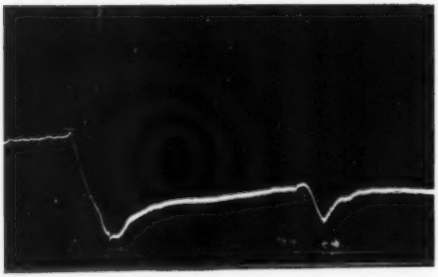

FIG. 4

Shows a fall of blood-pressure, the first part of which is due to paralysis of the vasoconstrictors; the second part is due to heart failure following respiratory paralysis. Artificial respiration restores the blood-pressure but *only* to 70 mm. Hg. Contrast with Fig. 1.

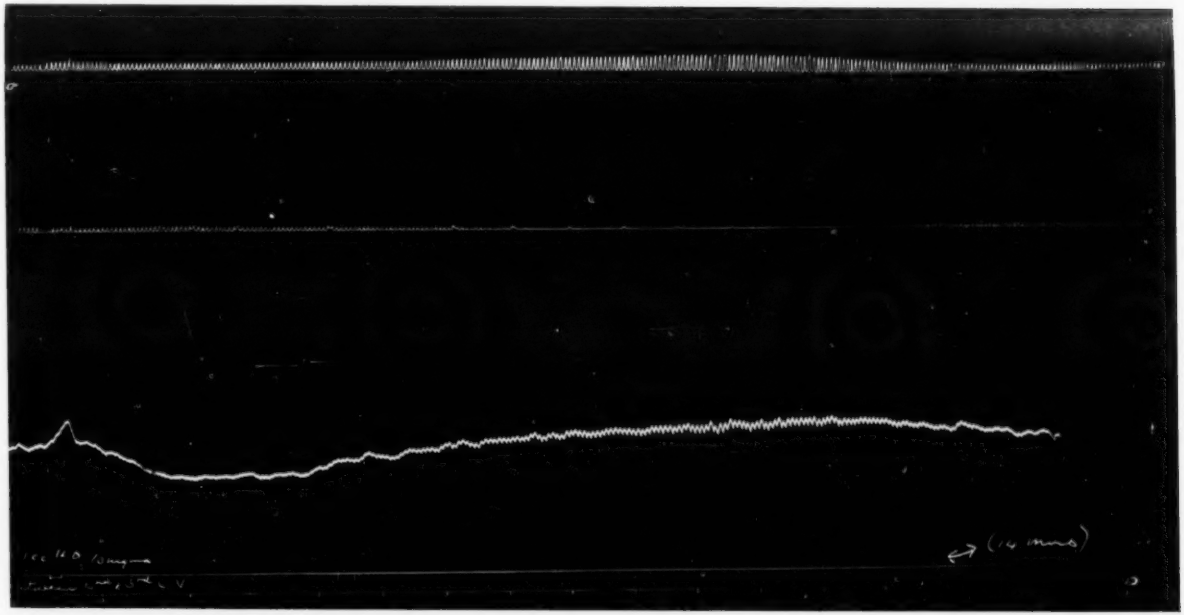

FIG. 5

Upper tracing, intercostal respiration. Middle tracing, diaphragmatic respiration. Lower tracing, blood-pressure. Shows a gradual paralysis of the diaphragm coincident with an enhanced intercostal activity, followed by a return of diaphragmatic respiration coincident with decreasing intercostal respiration.

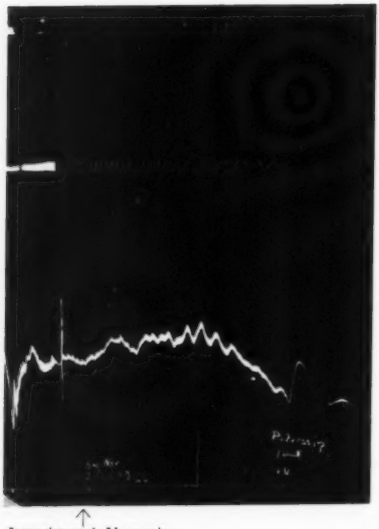

FIG. 8

Upper tracing, respiration. Lower tracing, blood-pressure. Shows a typical central paralysis as opposed to a phrenic. Respiration is slowed but not decreased in amplitude.

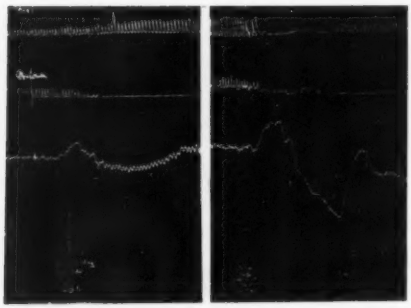

FIG. 9 FIG. 10

For explanation of these diagrams see text.

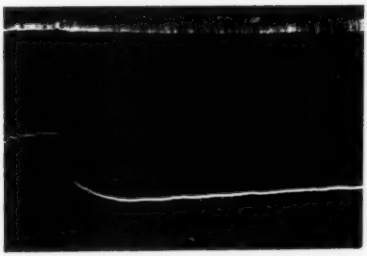

FIG. 11

A big fall of blood-pressure follows this injection of 50 mg. of Novocain into the lumbar theca, but a similar injection a few minutes later fails to increase the fall. (See text.)

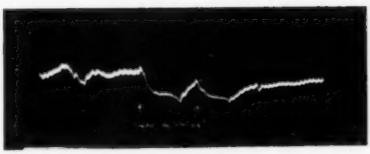

FIG. 12

The blood-pressure having been lowered to 60 mm. Hg by an intrathecal injection is reduced considerably by traction on the stomach. It rises on release, to fall again when the traction is renewed.

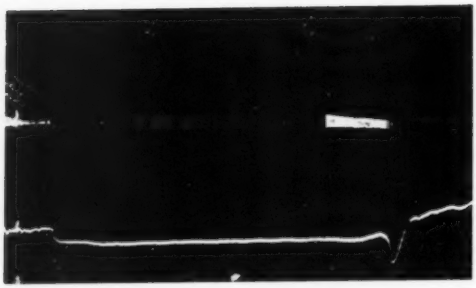

FIG. 15

For explanation of this diagram see text.

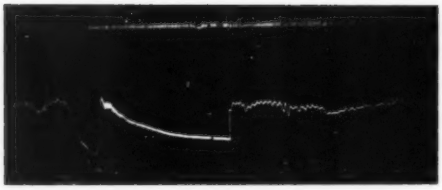

FIG. 16

10 mg. of Novocain paralysed the respiratory centre, a fatal fall of blood-pressure supervening. Rescue by artificial respiration. Later when the cat had completely recovered and was supporting life by its own respiration 1 ml. of saline was given intravenously. It had no effect on the blood-pressure or the respiratory tracing. A minute or two later 20 mg. of Novocain, or twice the dose which given intracisternally produced death in one and a half minutes, were given into the jugular vein. There is no effect discernible either on the respiratory tracing or on the blood-pressure.

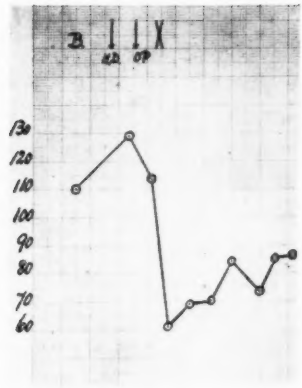

FIG. 17

B—Before the injections. HD—Heavy Duracain. OP—Operation. X—Exploration of the abdomen.
Notice the rise of blood-pressure after the injections of ephedrin and Durocain, the big fall immediately following the abdominal exploration, and the slow subsequent rise. Ordinates: blood-pressure. Abscissæ: 10-minute intervals.

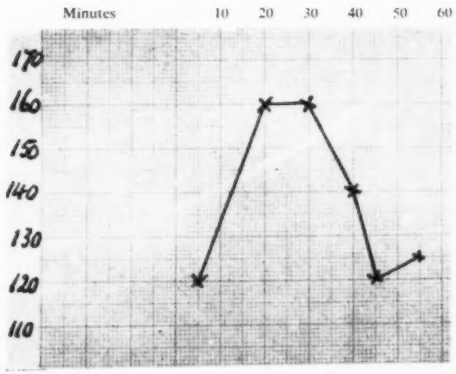

FIG. 18

The blood-pressure record of a man aged 45. The surgeon asked for a high anæsthesia as long as possible. The patient was given 300 mg., that is 3 ml., heavy Duracain. The operation lasted only three-quarters of an hour, but during the whole of that time the blood-pressure was as high or higher than it had been before the operation, even though the abdomen was fully explored and numerous adhesions round a previous gastro-jejunostomy were broken down. This patient did not feel towel clips put into the skin over either shoulder, showing that he was anæsthetized up to the level of the fourth cervical. He could move his arms, could breathe freely and had no fall of blood-pressure. It is difficult to suppose that anterior roots higher than the fourth dorsal were in any way affected by the anæsthetic—a difference of at least 8 segments.

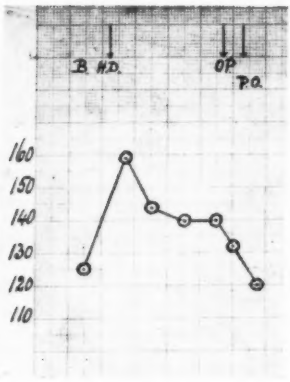

FIG. 19

Represents the blood-pressure in the case of an abdominal section to which the surgeon came half an hour late. Note that 33 minutes after the injection of the anæsthetic the blood-pressure was 15 mm. Hg higher than it was before the injection was given. After the opening of the peritoneum it sank below that level.

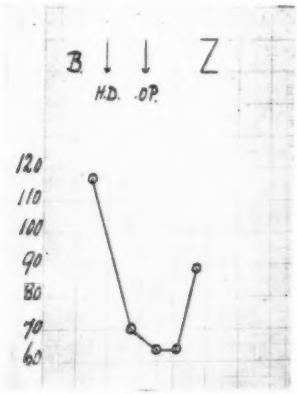

FIG. 21

Note immediate fall of blood-pressure following the injection of the anæsthetic solution, in a case of acute appendicitis with marked peritonitis. Accompanying the fall of blood-pressure the disappearance of the pain was equally dramatic.

He died 10 days later of uræmia. During the rescue operations a sample of blood was taken and examined, and found to contain 160 mg. per cent urea.

I was called to a nursing home one night to give a spinal anæsthetic, only to find that the surgeon had given the anæsthetic himself. The patient died 10 minutes later. The symptoms in this case had been more or less persistent vomiting; the diagnosis of intestinal obstruction had been made and, as subsequent events proved, was almost certainly wrong. It was probably another case of uræmia.

Two other cases have come my way, one of which was immediately fatal, and the other died a week later. Though the blood was not examined in these three latter cases, the symptoms in all were so alike and so characteristic that it would seem they had the same pathological basis. Two or three minutes after giving the spinal anæsthetic the patients seem as usual and then, suddenly—sometimes in the middle of a sentence, the patient becomes unconscious, and the physical collapse accompanies this. These cases are all surgical mistakes, cases of uræmia supposed to be intestinal obstructions; naturally they occur very seldom. No explanation has so far been forthcoming as to why or how they happen. If the C.S.F. combined with the anæsthetic to form a compound which had anæsthetic properties then the explanation would be simple; but no such compound is known. The symptoms suggest that a large overdose of the spinal anæsthetic has been given when in fact it has not.

DOES NOVOCAIN AFFECT THE HEART?

We have seen in Fig. 1 that a dose of 10 mg. Novocain injected into the cisterna magna causes death in a few minutes owing to cardiac failure, but obviously secondary to respiratory failure. Is the cardiac failure ever primary?

All the evidence that we have been able to adduce is against it.

The record shown in Fig. 15 suggests at first sight that the fall of blood-pressure was occasioned by a primary cardiac failure. Twenty-five mg. of Novocain were injected intrathecally in 0.5 ml. saline. A perfectly typical fall of blood-pressure took place to 60 mm. Hg. Half an hour later the blood-pressure began to rise slowly and slightly, following a slow steady fall in the amplitude of respiration. Six minutes later the blood-pressure fell to a fatal issue. Less than two minutes of artificial respiration saved the cat's life.

WHAT IS THE EXPLANATION OF THESE FACTS?

Why did the blood-pressure fall? Because the heart failed.

Why did the heart fail? Because it was called upon to work against a rising blood-pressure.

Why was the blood-pressure rising? Because of asphyxia due to slowness of respiration.

Why was the respiration slow? Because the cat had been overdosed with a barbiturate, and instead of responding by increased effort to the accumulated CO_2 it was poisoned by this CO_2.

How do we know that respiration had not been paralysed by the Novocain? Because of the small bulk of the injection, the time that had elapsed since giving it, and the fact that the vasomotor apparatus was active; and finally, and quite conclusively, by the effect of two minutes of artificial respiration. This washed out the CO_2, oxygenated the blood, raised the blood-pressure, and so enabled the heart which had failed against a blood-pressure of 60 mm. Hg

to work against a blood-pressure of 110 mm. Hg. This, then, was a cardiac failure secondary to respiratory failure and not a primary heart failure.

What of absorption of the anæsthetic into the blood as a cause of death on the table? Fig. 16 shows a typical central paralysis of respiration following the injection of 10 mg. of Novocain into the cisterna magna. The cat was rescued by artificial respiration, and after an interval of about an hour, when the cat had recovered completely from the effects of the intracisternal injection and had been maintaining itself by its own unaided respiration, 1 ml. of normal saline was injected into the jugular vein; this was followed a few minutes later by another injection of 1 ml. saline into the jugular vein, but this time it contained 20 mg. of Novocain. As far as could be ascertained by the respiratory and blood-pressure curves neither injection made any difference to the cat. That is to say that an injection twice as strong, when given intravenously, as the one which, when given intracisternally, had killed the cat, but for rescue, in one-and-a-half minutes, had not the slightest effect.

In order to ascertain exactly how much Novocain was required to bring about a fatal result, we injected 7.5 mg. of Novocain per minute intravenously, and it took 24 minutes before respiration was paralysed. The intravenous dose required to paralyse respiration when given at the rate of 7.5 mg. per minute is then 180 mg. Even with this enormous dose artificial respiration restored the cat's own respiratory activity in two minutes. As artificial respiration saved the animal within two minutes, the question of the collapse of the vasomotor apparatus or the heart did not arise. It is obvious that still larger doses would be required to prevent the functioning of these two vital mechanisms.

Of late, without causing any collapse, Novocain and Nupercain have been injected intravenously in the human subject in larger doses than any that could be absorbed from the theca in the course of even a long operation under spinal anæsthesia.

CEREBRAL ANAEMIA

Labat (1928) stresses cerebral anæmia as the cause of death and gives two diagrams in his paper; one of a patient in the Trendelenburg position which he labels " Life ", and one in the Fowler position which he labels " Death ". Important as the Trendelenburg position undoubtedly is, especially when the blood-pressure is low, it is quite certain that this position would not save the patient's life if there were sufficient anæsthetic on the respiratory centre to paralyse it; for then, though there would be plenty of blood in the brain and the medulla, it would not be oxygenated and that would be fatal. Labat's technique of putting the patient into the Trendelenburg position immediately he has given the anæsthetic tends to put the anæsthetic on to the centre and to bring about that condition of getting the anæsthetic " too high " which we are so anxious to avoid. That he has escaped this disaster must be due to the fact that he has invariably used doses insufficient to paralyse the centre, and not to the Trendelenburg position.

THE MECHANISM OF DEATH

We have seen in the course of these experiments that death is always due to heart failure and yet there is no evidence to indicate that the spinal anæsthetic has any effect on the heart; that is to say, the heart failure is never primary, always secondary, to failure elsewhere. The

activity of the heart muscle depends upon its oxygen supply. This oxygen, which is carried to it by the blood in the coronary arteries, depends as regards quantity on the minute volume of the circulation and the oxygen tension in that blood. How, then, does a spinal anæsthetic interfere with the oxygen tension of the blood in the coronary arteries and the volume of that blood per minute?

The only way, apparently, that the anæsthetic can interfere is by paralysing respiration, either centre or phrenics. Even so, paralysis of the phrenics would not be sufficient in an animal that is healthy unless the intercostals were paralysed too. In ordinary clinical medicine where the injection is made in the lumbar region, if the phrenics were paralysed the intercostals would be so too, but if for any purpose an injection were made, say in Jonesco's (1919) *medio cervicale* position, the phrenics might be paralysed and the intercostals unaffected by the anæsthetic as shown in Fig. 5.

If, then, the only way in which the anæsthetic can interfere with the oxygen tension in the blood and the minute volume of the circulation is by paralysing respiration, can we say that artificial respiration will always save the patient? If the patient is a cat in the pharmacological laboratory the answer is " Yes ". Unfortunately we cannot be quite so dogmatic when the patient is a human being, even though, if artificial respiration is applied in time, there will be few disasters. In clinical practice, if correct technique is used, respiration (phrenic or centre) never should be paralysed.

Though the only way in which a spinal anæsthetic can interfere with the oxygen tension in the blood and the minute volume of the circulation directly is by paralysing respiration, if that should never occur, can the anæsthetic

be responsible for the patient's death? The answer to this question is that it can be a factor only in the death of a patient who comes into the category of "a grave risk". How does it act in such a case? If by reason of toxaemia (diphtheria, influenza), fatty infiltration (obesity), myocardial degeneration (old age, arterio-sclerosis), the heart is feeble we have a "grave risk". To this patient is given a sedative, morphia and atropine or Omnopon and Scopolamine, followed an hour later by the spinal anæsthetic; then the blood-pressure falls, owing to the lack of vigour of the patient; those vessels not affected by the anæsthetic, which usually contract and keep up the blood-pressure, fail to act; the medulla is depressed by the sedative; the bases of the lungs, owing to the Trendelenburg position, expand inadequately; the blood therein is not oxygenated; afferent impulses normally stimulating respiration are depressed, and absent in the lower two-thirds of the body. Every time the blood circulates these conditions tend to get worse. The feeble heart, supplied with anoxaemic blood, passes this on to the medulla already depressed by a sedative and, as Sebrechts (1930) puts it, "Les choses se passent comme si tout organisme vivait 'au ralenti' dans une espèce de léthargie qui s'étend déjâ aux fonctions bulbaires". The depressed medulla, the partially inflated lungs, the low blood-pressure, the abolished reflexes, the feeble heart, all tend to anoxaemia, and there comes a time when there is not sufficient oxygen in the coronary arteries for the cardiac muscle cells to carry on, and then the heart fails.

Again, in cases of haemorrhage or where for any reason the fluid in the circulating system is diminished, as in shock, there is a compensatory vasoconstriction to conserve what blood there is for the vital organs. A spinal anæsthetic, given before the fluid has been restored, may so spread the

circulatory bed that there is not enough blood for heart, brain, and lungs. Here, then, the minute volume to the coronary arteries would be insufficient and the heart would fail.

THE TECHNIQUE

The technique which emanates from these experiments emphasizes the following points:

(1) Use a solution heavier than C.S.F., because, if by mistake too big a dose has been injected, the brunt of the attack falls on posterior roots, which is of little consequence. If light solutions are used in excess the anterior roots controlling blood-pressure and breathing are affected.

(2) Give atropine and morphia rather than Omnopon and Scopolamine, as the latter are too depressing to the respiration.

(3) Give oxygen through the nose throughout the operation.

(4) Put the patient in the Trendelenburg position ten minutes after you have given the injection.

(5) If the patient is dehydrated do not give the anæsthetic until the deficit has been made good.

If the mechanism of death is understood, and the technique here set out is used, there should be no deaths under spinal anæsthesia. If mistakes are made, such as operations on a uræmic patient in mistake for intestinal obstruction, or desperate risks (perhaps justifiable) are taken, such as amputation of a leg for gangrene in a moribund diabetic, a death on the table is likely to happen occasionally but can scarcely be put down to the anæsthetic.

Figs. 1, 2, 5, 9 and 10 are reprinted from the *Journal of Pharmacology and Experimental Therapeutics*, by kind permission of the Editor.

REFERENCES

Dallemagne, Marcel J. (1949), *Aspects-actuels de L'anesthesiologie*, Masson, Paris.

Forgue, E., and Basset, A. (1930), *La Rachianesthesie*, Masson, Paris.

Henderson, V. E., and Johnston, J. F. A. (1932), *Curr. Res. Anæsth.*, **11**, 78.

Jones, W. Howard (1931), *Brit. med. J.*, **2**, 488.

Jonesco, T. (1919), *La Rachianesthesie Generale*, Masson, Paris.

Koster, H., and Kasman, L. P. (1929), *Surg. Gynec. Obstet.*, **49**, 617.

Labat, G. (1928), *Amer. J. Surg.*, **5**, 625.

Maxson, L. H. (1938), *Spinal Anæsthesia*, Lippincott, London.

Pannett, C. A. (1933), *Lancet*, **2**, 169.

Sebrechts, J. B. (1930), *Bull. Acad. Med. Belg.*, **10**, 543.

Sebrechts, J. B. (1934), *Brit. J. Anæsth.*, **12**, 4.

Seevers, M. H., and Waters, R. M. (1932), *Curr. Res. Anæsth.*, **11**, 85.

PHARMACOLOGICAL EFFECTS OF PETHIDINE ON THE LARYNX SEEN DURING INTUBATION

By HENNING RUBEN *and* A. KLOCK ANDREASSEN

STIMULATED by the reports of the good results of using pethidine during anæsthesia (Mushin and Rendill-Baker, 1949; Neff Mayer and Thompson, 1950; Brotman and Cullen, 1949), we not only introduced this drug into our routine practice but have investigated its use as an alternative to the barbiturates for intubating the larynx.

This paper describes our observations of some of the pharmacological actions of pethidine on the larynx during the use of this drug for endotracheal intubation. From a structural point of view pethidine (Demerol, Isonipecaine) is in some ways similar to morphine and in others to atropine (Yonkman, 1948). Like morphine it has a piperidine ring (1-methyl-4-phenylpiperidine-4-carboxylic acid), and this probably accounts for its analgesic effects. However, on the other hand, like atropine it has both antihistaminic and anticholinergic properties.

The immediate effect of pethidine administered intravenously to the anæsthetized dog is a marked hypotension of rather short duration. Like most of the anæsthetics it is also depressant to the respiration. Sedation, however, is not as marked as with morphine. The degree of respiratory and circulatory depression is dependent on the rate of injection of the drug. Slow injection largely eliminates these undesirable effects.

The difficulties attending endotracheal intubation are nearly all due to the laryngeal reflexes. To obtund these

reflexes requires either deep general anæsthesia, surface anæsthesia of the larynx or the use of relaxants. All these various methods have been recommended at one time or another for intubation. Diethylether or cyclopropane, for example, are widely used for this purpose, but the attainment of the proper degree of anæsthesia for laryngoscopy with these anæsthetics may be rather time-consuming. Other methods which are quicker are now coming into general use. An example of this is the combination of short acting barbiturate injected intravenously, combined with surface anæsthesia or curare. Such methods, however, are apt to produce profound respiratory depression or even apnœa, and the intubation may well become a major procedure compared with the rest of the anæsthesia. In the hand of a tyro or if the dosage is improperly judged, the risk of laryngospasm with the possibility of hypoxia may be a very real menace to the patient in these circumstances.

While using pethidine as a supplementary agent to N_2O/O_2 anæsthesia we got the impression that pethidine by itself possessed a selective depressant effect on the laryngeal reflex. We therefore substituted pethidine for thiopentone in combination with a relaxant and tried this new combination for intubation. The result of this change we found noteworthy. In addition, this combination proved to be useful for such a purpose as oesophagoscopy, in which it gave good results in a few cases.

Before discussing the effects of pethidine on the larynx we feel it necessary to describe in some detail how the drug was administered.

TECHNIQUE

A 1 per cent solution of pethidine in physiological saline was injected intravenously with a speed of about 60 mg.

per minute. The patients were premedicated with moderate doses of morphine and scopolamine about 75 minutes beforehand. In some cases no premedication was used and no alteration in results was noticed. It was found that pethidine almost obtunded the pharyngeal and laryngeal reflexes without the necessity of inducing a deep plane of anæsthesia, and without the necessity of hurrying the intubation.

About 50–80 mg. of pethidine are injected intravenously and the administration of 80 per cent nitrous oxide and 20 per cent oxygen is started. As soon as the patient is asleep, or almost so, 5–10 mg. tubarine or 30–60 mg. Flaxedil are injected. After a pause of one minute, a further dose of about 50 mg. pethidine is injected. After this, a gentle attempt is made by touching the epiglottis with the forefinger or by inserting the laryngoscope. Usually the patient will tolerate this, and the intubation is then performed. If he does not tolerate the laryngoscope or finger, a further dose of pethidine has to be given.

Stridor or laryngeal spasm has not been encountered in this series of intubations. Coughing has been an unusual occurrence. When it has happened, it has been limited to a few feeble coughs. Often the cords are seen to be widely abducted. If touched by the tube, they sometimes adduct, but usually do not. They are not seen to be in spasm, as often happens when the barbiturates are used. As soon as the tube is removed from the cords, they again abduct. With the dosages we give, this type of reaction remains for at least five minutes. The technique described has now been used in 189 cases.

In order to discover whether pethidine depressed the laryngeal and pharyngeal reflexes in a selective manner, a study of 25 consecutive cases was made in which pethidine

was used as the sole agent for intubation. In 2 cases a dose of 40 mg. Flaxedil was used in addition, not for the abolition of the reflexes but to increase relaxation of the jaws. No attempt was made to choose the patients. Of the 25 cases, 19 were between 30 and 70 years of age; the youngest patient was 7, and the oldest was 82 years of age. The doses of pethidine for the performance of the intubation are given in Table I. Even for robust adults a total dose of 200 mg. was not exceeded. The results of this study are given in Table II. In addition to the effect on the laryngeal and pharyngeal reflexes we have found that immediately after the injection a slight relaxation of short duration of the masseters was noticed, only a small resistance being felt when the jaw was lifted with the laryngoscope.

TABLE I

Drugs used.

(*a*) *Premedication, mg. morphia/mg. scopolamine.*

	0/0.	5/0.2	7.5/0.3	10./0.4	12.5/0.5	15/0.6
No. of cases	6	3	1	13	1	1

Total 25.

(*b*) *Pethidine, mg.*

	40	60	90	100	120	140	150	160	180	200
No. of cases	1	1	1	5	4	2	1	2	2	6

Total 25.

(*c*) *Cases without premedication, pethidine mg.*

	100	120	160	180	200
No. of cases	1	1	1	1	2

Total 6.

TABLE II

Pethidine effect on reflexes.
No. of patients: 25.

		No. of cases	Total
Larynx—Spasm		0	
	Coughing	6	25
	No reaction	19	
Pharynx—Swallowing		4	
	No reaction	21	25

In addition, the pharyngeal musculature seemed to be a little relaxed. The patient usually was awake and co-operative, but a little slow in mental reaction. When asked to open his mouth, he did so. A few of the patients were lightly asleep.

The disadvantage of pethidine for intubation seems to be the respiratory depression. Sometimes a respiratory rate of about 6–8 per minute occurred immediately after the intubation. This depression lasted a very few minutes, after which the respiratory rate again became more than 12. In fact, in the last part of this series, we took a slight slowing of the rate of respiration as a guide that sufficient pethidine had been given for the performance of the intubation. When the respiratory rate was about 10 per minute, the dosage of pethidine was generally sufficient for intubation to be attempted. Apnœa was not seen in our series, nor was any other complication apart from respiratory depression.

The techniques described here were used for the induction of several kinds of anæsthesia, including ether, cyclopropane, trichlorethylene, and N_2O/O_2 pethidine. We have found that the respiratory depression was of such short duration, that it had very little disturbing effect on the maintenance of anæsthesia by any of the inhalation agents mentioned.

SUMMARY AND CONCLUSION

In a series of 189 patients pethidine was used for endotracheal intubation. The technique used is described.

Certain pharmacological effects of pethidine on the larynx were found.

With the doses used there was a marked depression of the laryngeal reflexes. On irritation, the vocal cords, in some cases, did not react; in others the cords adducted but as soon as the irritating stimulus was removed they abducted again. No laryngospasm was seen. Very seldom a few weak coughs were encountered after the intubation. These effects of pethidine seem to us to make the act of intubation a lesser hazard to the patient. A further advantage was that when depression and almost abolition of the reflexes was obtained with pethidine the patient was still only lightly asleep. This is contrary to what we find when general anæsthetics are used for the same purpose.

Because of these results we believe that the use of pethidine combined with N_2O/O_2 and a relaxant may prove a useful technique for intubation where, for some reason, a quick induction is desired, and where the use of barbiturates is regarded as contra-indicated.

REFERENCES

Brotman, M., and Cullen, Stuart C. (1949), *Anesthesiology*, **10**, 696.

Mushin, W. W., and Rendill-Baker, L. (1949), *Brit. med. J.*, **2**, 472.

Neff, W., Mayer, E. C., and Thompson, R. (1950), *Brit. med. J.*, **1**, 1400.

Yonkman, F. F. (1948), *Annals of the New York Academy of Sciences*, **51**, 59.

GALLAMINE IN THE DIAGNOSIS OF MYASTHENIA GRAVIS

By JOHN W. DUNDEE

SINCE gallamine (Flaxedil) was introduced as a muscle relaxant in anæsthesia two years ago, only one case of sensitivity to it has been reported (Fairley, 1950). Its action is similar to that of d-tubocurarine chloride, interfering with the chemical mediator at the myoneural junction (Mushin, Wien, Mason and Langston, 1949; Wilson and Gordon, 1949). Since sensitivity to d-tubocurarine chloride is a recognized clinical entity (Gray and Halton, 1948), it is to be expected that a similar condition exists with gallamine. This possibility is borne in mind when myasthenia gravis is listed as an absolute contra-indication to its use (Harrison, 1950).

Increased sensitivity to curare has been used as a diagnostic test in suspected myasthenia gravis, but is not recommended as a standard procedure (Horder, 1950). One-tenth to one-fortieth of a mild curarising dose is used (Eaton, 1947), the response varying with the severity of the disease. The sparing effect of gallamine on respiration, as compared with d-tubocurarine chloride, may well make it a safer drug for this test. Both drugs were administered to the following patient and the responses compared.

A woman, aged 31, had been investigated in several hospitals for periodic loss of voice and difficulty in lifting her head from the pillow. Intramuscular injection of neostigmine 1.5 mg. and atropine 0.65 mg. produced no improvement in her condition. Complete ptosis, a choking sensation

and troublesome salivation followed intravenous injection of 1 mg. d-tubocurarine chloride. This response appeared 50 seconds after injection, and was immediately reversed by neostigmine 1.5 mg. and atropine 0.65 mg. The patient described the procedure as definitely unpleasant.

Two days later 1.5 mg. gallamine intravenously produced complete ptosis and loss of voice, but no choking sensation or excessive salivation. The maximum effect occurred 25 seconds after injection and was immediately reversed by neostigmine and atropine. From the patient's point of view this test was much less unpleasant than the previous one and, after the antidote had taken effect, she felt stronger than she had been for weeks.

Most observers have reported a more rapid onset of action with gallamine than with d-tubocurarine chloride. This is borne out in this case. The patient's weight was 60 kg. (9 stone 7 lbs.), the doses administered being d-tubocurarine chloride 0.016 mg./kg. and gallamine 0.025 mg./kg. The more severe response to d-tubocurarine chloride may have been due to the relatively large dose. Assuming 15 mg. as a mild curarising dose, the 1 mg. she received was not expected to be excessive, in view of the mildness of her symptoms. The potency of gallamine is about one fifth that of tubocurarine and yet 1.5 mg. produced a response of as great a magnitude, without the distressing subjective sensations, as did the d-tubocurarine chloride.

From this case it can be seen that gallamine in doses of 0.025 mg./kg. is as reliable a test for the diagnosis of myasthenia gravis as is d-tubocurarine chloride. In this dosage it is not unpleasant to the patient. In spite of the ptosis there was no respiratory distress and no obvious intercostal paralysis. Nevertheless it would not be con-

sidered advisable to perform the test without having adequate means available for inflating the patient with oxygen, and syringes containing neostigmine and atropine ready to hand.

Lest there be any doubt about the diagnostic value of gallamine, it was administered in the same dosage as above to a proven case of myasthenia gravis. The therapeutic neostigmine which the patient was receiving had been omitted for 24 hours. The response was again very dramatic and there were no unpleasant effects. Once again the patient described the sensations as less distressing than after the diagnostic dose of d-tubocurarine chloride which had been given several months previously. The response to both muscle relaxants was more definite than had occurred after intramuscular neostigmine and atropine (Warner, 1950). Two further cases have been tested since with similar results.

The superiority of gallamine and d-tubocurarine chloride over intramuscular neostigmine is shown by these cases. Present experience suggests that gallamine is a less unpleasant drug from the patient's point of view.

REFERENCES

Eaton, L. M. (1947), *Proc. Mayo Clin.*, **22**, 4.
Fairley, H. B. (1950), *Brit. med. J.*, **2**, 986.
Gray, T. C. and Halton, J. (1948), *Brit. med. J.*, **1**, 784.
Harrison, B. (1950), *Anæsthesia*, **5**, 100.
Horder, Lord (1950), *British Encyclopædia of Medical Practice, Cumulative Supplement*, **9**, 309, Butterworth, London.
Mushin, W. W., Wien, R., Mason, D. F. J., and Langston, G. T. (1949), *Lancet*, **1**, 726.
Warner, E. C. (1950), *Saville's System of Clinical Medicine*, 13th edition, p. 1005, Arnold, London.
Wilson, H. B., and Gorden, H. E. (1949), *Lancet*, **2**, 504.

ANÆSTHESIA IN URUGUAY

By RONALD F. WOOLMER

URUGUAY is an independent republic occupying an important position on the eastern seaboard of South America. Though it is dwarfed in size by its neighbours, Brazil and Argentina, its social, cultural and scientific development is well to the fore. The Uruguayans do not believe in revolutions, and the country's political and economic stability, among the unrest which characterises so many South American republics, shines like a good deed in a naughty world.

The medical profession is highly regarded in Uruguay; and the Faculty of Medicine in the University of Montevideo—which serves the entire country—is an up-to-date, well housed and progressive institution. In former times it drew its inspiration largely from France, but nowadays it looks mainly to Britain and the U.S.A., and many lecturers from these countries visit Montevideo.

Until five or six years ago, anæsthesia had no place in the medical curriculum, and standards were as low as anywhere on the European continent. Some years ago, however, one or two able practitioners in Montevideo began to devote their energies to the study and practice of this branch of medicine. Their efforts quickly achieved recognition, and the need to further the study of anæsthesia in Uruguay and to include it in the medical curriculum soon became apparent. Accordingly, the Faculty of Medicine in the University decided to invite, from Britain,

someone who would teach modern methods of anæsthesia in the University, which, in time, would be able to supply a flow of trained anæsthetists for the whole republic.

The writer arrived in Montevideo at the end of July. This is the beginning of the Uruguayan winter, and the swiftness of air travel made the transition unpleasantly abrupt, though in this latitude, which is the same as that of Capetown, winter is less severe than in Britain. The British Council had been an effective intermediary in negotiations between the two countries, and its Chief Representative in Uruguay, Mr. J. Bruton, who was in close touch with the University authorities, was of the greatest help throughout my visit.

I spent the first few days getting to know the University authorities, and the anæsthetists and surgeons with whom I was to work for the next two or three months. This was a most agreeable process, for the Uruguayans, with their warm-hearted friendliness and extroverted Latin temperament, are the gayest and most hospitable people imaginable, and they took endless trouble to make me feel at home. They combine their light-heartedness, however, with a formidable capacity for hard work. Hospital lists start at 7.30 or 8.0 in the morning, and private cases an hour earlier. Hospital operating is nominally confined to the forenoon, but lists often continue until well into the afternoon, and there is generally an hour or two of ward visits or private work in the early evening.

A course of lectures was arranged at 7 o'clock in the evenings, as being the only time at which the majority of anæsthetists would be free; and in spite of their many commitments, attendance at these lectures was near the maximum throughout. They were often followed by lively discussions which might be continued during dinner, a meal

which in Montevideo may be taken at any time between 9.0 p.m. and midnight.

All types of surgery are performed in Montevideo, and there are a large number of hospitals. The larger hospitals are old and decrepit in the extreme, and the good work which is done in them is carried on under considerable difficulties. They are shortly to be augmented or replaced by a gigantic new hospital, which stands, white and elephantine, in a commanding position overlooking the football stadium. Though it is structurally complete, it is not yet fully equipped, and no-one can forecast when it will be ready to receive patients. The operating floor is near the top of the building, and from the windows of its fifteen theatres a magnificent view is afforded of the city with its parks and squares, and of the coast and estuary with its numerous sandy beaches and its groves of pine and eucalyptus trees. The operating rooms are neatly planned, mostly in pairs, with a well placed spectators' gallery over each. Communication with the gallery is provided by microphone and loud speaker, and one of the theatres is equipped for television. Each theatre has an anæsthetic room, which is a rarity in the hospitals of Montevideo. Unfortunately, however, the effect of this is ruined by the fact that the outgoing patient has to be wheeled through the anæsthetic room on his way back to the ward. There is no provision, either, in this vast hospital, for central direction of the anæsthetic services or any accommodation for the storage of spare anæsthetic equipment. This is not surprising, considering that the hospital was designed twenty years ago; but plans have now been made to meet these deficiencies.

The standard of surgery in Montevideo is high, though specialization is not carried to the same degree as in this

country. The first Blalock's operation was performed before my arrival, and the second while I was there. Hydatid disease is extremely prevalent in Uruguay, and " hydatid cyst of lung " appears several times a week on the general operating lists, so that nearly all the anæsthetists have some experience of this relatively simple type of lung surgery.

In the city of Montevideo there are about 25 anæsthetists. For a town of a million inhabitants, in which the surgery is done in a large number of scattered hospitals, this is less than sufficient. Though none has been specializing for more than four or five years, the best of the anæsthetists have attained a high degree of technical skill, and their theoretical knowledge is up-to-date. Not all of the anæsthetists are qualified medical practitioners. This is difficult for a visitor from Britain to understand at first. In Uruguay there is no limit to the time which may be taken over qualification. Though the curriculum may be covered in about five years, six or seven is usual, and even eleven or twelve is not unknown. Many students have no financial resources, and have to engage in some side-line in order to keep themselves and their families. With the increasing realization, by surgeons, of the benefit to be gained from professional anæsthetists, there is a considerable demand for the services of all those, be they undergraduates or postgraduates, who have acquired the necessary knowledge and technical skill. The result is that there are a number of students, in what corresponds to our fifth year, who are so busy giving anæsthetics both for regular hospital lists and for private cases, that they have little time left to press on with their studies and achieve qualification. This is of course a thoroughly bad principle, and must be harmful in the long run, though it should in fairness be

said that most of these men are intelligent, enthusiastic and hard working, have acquired a high degree of knowledge and skill, and are determined to finish their medical studies as soon as they can.

There is yet some way to go, in Uruguay, in the recognition of anæsthesia as a speciality. Some of the older surgeons still rely on conduction analgesia given by themselves. The standard of such analgesia is low. The " cut—wince—inject " technique is too much in evidence, and spinals are given with little understanding of the underlying principles.

The aspect of anæsthesia in which the country is least advanced is the attitude adopted to the patient. Children's tonsils are still removed without any anæsthetic, and far too little attention is given to sparing the feelings of adult patients. Obstetric analgesia is almost unknown, and the pain of labour is regarded as too slight a thing to be treated seriously.

The standard equipment in many of the hospitals is the Ombredanne apparatus, which is regarded as a suitable implement for the student or nurse anæsthetist; but the majority of hospitals which are visited by professional anæsthetists have more elaborate equipment. The Foregger machine, with water depression type flowmeters, is most in evidence, but a few McKessons and Heidbrinks are also to be seen. It is hoped that some British equipment will be available before long. Cuffed Magill's tubes are scarce, and effective pharyngeal packing is not often used, so that in many cases it is difficult to achieve a really airtight fit between the patient and the machine. Nitrous oxide is imported from the United States, so that it is about as expensive as cyclopropane, and has to be used as sparingly. For this reason semi-closed gas-oxygen-ether—the stand-

by of the semi-skilled—is not often used, and even the fledgeling anæsthetist must accustom himself in the use of the closed circuit. Muscular relaxants have been in regular use for several years; they are used with understanding and the doses are never excessive.

On the whole, anæsthesia is a thriving and advancing speciality in Montevideo. In the rest of the republic, which is sparsely populated and has no big towns, it is still primitive; and its development there must be slow for economic reasons.

A striking thing to the visitor is the apparent haphazardness of the arrangements for anæsthesia. This is due partly to the very rapid growth of the speciality, and partly to the Latin temperament with its dislike of regimentation. There is little co-ordination of the hospital anæsthetic services, anæsthesia is not included in the undergraduate teaching curriculum, and there are as yet no satisfactory facilities for postgraduate teaching or for research. The faculty of Medicine is very much alive to these deficiencies (which indeed are not peculiar to Uruguay), and one of my tasks was to draw up a plan, with the authorities, for establishing the speciality on a firmer basis. This plan will have to be put into operation in stages. The first stage will be the systematic instruction of undergraduates and the provision for would-be specialists of a proper groundwork in the basic sciences. Next will come their further instruction in the principles and practice of clinical anæsthesia; but this part of the plan cannot mature until suitably qualified instructors are available. It is hoped that a number of Uruguayan anæsthetists may be able to spend some time studying at the various centres in Britain, so as to fit themselves for teaching more advanced anæsthesia in their own country.

The two and a half months I spent in this attractive and

vigorous country (where there is no such thing as income tax) were sufficient to convince me that it will not be many years before the standard of anæsthesia equals the best to be found in Europe. The anæsthetists are all young, enthusiastic and eager to learn; and their efforts to improve their skill have the whole-hearted support of the surgeons. They recognize that they have something to learn from British methods of anæsthesia. We in Britain would be the happier if we could adopt some of the many pleasant ideas which form part of the Uruguayan way of life.

Dr. Magill

IVAN MAGILL

By STANLEY ROWBOTHAM

IVAN WHITESIDE MAGILL was born at Larne, Co. Antrim, on the 23rd July, 1888, and the early years of his life were spent in Ireland. He was educated at Larne Grammar School and Queen's University, Belfast. From here he graduated M.B., B.Ch., B.A.O., in 1913. Immediately after qualification he came to England, taking the posts of House Surgeon and later of R.M.O. at Liverpool Stanley Hospital. The following year he became R.M.O. at Walton Hospital, in Liverpool, but this post he held for only six months; for in August the Great War burst upon the world, and as with so many young men at this time of confusion and unrest, it profoundly altered Magill's career: but for the war it is unlikely that his inclinations would have been for anæsthesia; at that time he had a decided predilection for surgery.

In 1915, Magill took a temporary Commission in the R.A.M.C., and in this Corps he served, with the rank of Captain, throughout the remainder of the war. During much of his service he acted as Medical Officer in charge of troops, and he was Medical Officer to the Irish Guards throughout the battle of Loos.

After the cessation of hostilities Magill became anæsthetist at Barnet War Hospital, and early in 1919 he was posted with the writer to the Queen's Hospital for Facial and Jaw Injuries at Sidcup. However much his experience at Barnet had taught him, anæsthetics for the newly developed art of plastic surgery presented problems which

might well have caused a man of lesser courage to think twice before he tackled them. Most of the anæsthetists at Sidcup were non-resident civilians, the newly posted officers were resident, and so it came about that they were precipitously and without training plunged into the task of administering some of the most difficult and hazardous anæsthetics that one could meet. These cases, however, not only intrigued Magill, but stimulated in him an obstinate tenacity, the result perhaps of his northern Irish descent. He threw all his energies into the problems concerned with the administration of the anæsthetics with which he was daily dealing, with what result the history of anæsthesia from 1920 onward bears eloquent witness. This, then, was the start of Magill's real life's work. Much was lacking in the anæsthetic techniques in use at Sidcup, and as realization of what was needed gradually came to him, he set about first the task of making himself a good anæsthetist, and then of improving and perfecting the methods in use and of designing new ones; it was in fact the genesis of a new era in anæsthesia.

When Magill joined the Hospital at Sidcup, the usual anæsthetic for plastic and jaw operations was rectal oil ether. This was far from being a satisfactory method; the patient, too light at the start, often became very deep during the operation; after return to the ward the recovery period was prolonged, sometimes as long as twenty-four hours. Moreover, given the occasional case where it was possible to maintain an even degree of anæsthesia throughout the operation, the problem of keeping an airway was always present, and often severely hampered the surgeon in his work. Oral airways and Silk's nasal tubes were commonly in use to meet this difficulty. Kelly's method of intratracheal insufflation had lately been introduced into

London by Shipway, and was in process of trial at Sidcup. The catheter was, for oral operations, passed through the nose, being viewed in the pharynx by a laryngoscope and guided into the larynx with forceps. For this purpose Magill invented a special forceps which have in fact found many other uses besides the one for which he originally intended them. Kelly's method proved to be an advance on rectal ether, but the amount of anæsthetic which the surgeon was forced to inhale and the bubbling of blood in the mouth caused by the returning air stream rendered it still far from an ideal procedure. Return airway tubes were accordingly introduced and for this purpose Silk's tubes were at hand, and often worked admirably. There then followed the discovery that the tube when passed down the nose sometimes actually entered the larynx, and that it could be nearly always manipulated so that it would do so. This led directly to the elaboration of the technique of blind intubation, a technique which Magill carefully worked out and described. It was from this time that the development of endotracheal intubation as we know it today originated; an advance in which Magill took an important and original part.

It was at once obvious that whereas endotracheal insufflation had many drawbacks, intubation of the larynx with a single wide bore tube had great advantages. It was, for instance, possible to give a pure mixture of gas and oxygen, a method which was both wasteful and difficult by insufflation. According to the type of operation it was desirable to introduce the tube via the mouth or via the nose. The latter method particularly intrigued Magill and when he discovered that the tube could be introduced "blind", without the use of a laryngoscope, he concentrated on this method, which he described to a meeting of

the Anæsthetic Section of the Royal Society of Medicine in 1928. Up to this time the tubes used for intubation were either Silk's nasal airway tubes, which were often too small, or home-made ones fashioned out of any curved piece of rubber tube that was available. Magill persuaded the manufacturers to mould a series of tubes in graduated sizes; this refinement enabled blind intubation to be firmly established as a technique to be used in every type of case and every age of patient where the nose was available for the tube.

Magill's career as an anæsthetist has been largely associated with the development of endotracheal anæsthesia, and he has invented and perfected many instruments and apparatus for use in this type of anæsthesia. He was, for example, the first to devise a laryngoscope with self-contained battery, and after his appointment to Brompton Hospital in 1923, he introduced methods which it is no exaggeration to say have revolutionized the anæsthesia, and as a direct result, the surgery, of chest operations. In 1931, he first used a catheter with an inflatable cuff to control the secretions in the operation of lobectomy, and in 1936 he produced his ingenious tubes, with instruments for their introduction, for one lung anæsthesia.

Throughout his career Magill has been associated with plastic surgery. He was probably the first to adopt the endotracheal technique for cleft palate and hare lip operations in infants. He still remains an outstanding exponent of this method, which has been copied by many anæsthetists throughout the country.

In 1924 Magill was appointed Anæsthetist to Westminster Hospital. He is now Senior Anæsthetist to the Hospital and has served on the Staff for 26 years. Although known largely for his work in endotracheal anæsthesia, he

has not been backward in other directions where invention and improvement of anæsthetic apparatus seemed necessary to him. As early as 1928 he adopted dry flowmeters for the measurement of gases, and in 1932 he suggested to Mr. A. C. King the adoption of rotameters for this purpose. They were at that time made in Germany and the cost was prohibitive; moreover, delivery could not be obtained under six months. It was not therefore until 1935 that he was able to obtain rotameters made to his own measurements in this country.

Basal anæsthesia was early adopted by Magill, and in 1930 he was responsible for introducing Nembutal from the U.S.A.

An incident worth recording which shows how advanced an outlook in anæsthesia Magill has always held comes from Dr. Harry Daly, who just before he left this country wrote: " It is interesting to note that while in London in 1935, Dr. I. W. Magill remarked to me that curare as a muscular relaxant might be used in place of spinal analgesia then under trial for thoracic surgery ".

When Magill first began his career as an anæsthetist there was but one established venue where anæsthetists met to discuss their problems and exchange experiences, this was the Section of Anæsthetics at the Royal Society of Medicine, which had developed in 1908 from the old Society of Anæsthetists. In 1931 Magill put before the Council of the Section the advantages which would accrue from a Diploma in Anæsthesia. The discussion on this project was recorded by him in the minutes on 6th November 1931, for he was at that time Senior Secretary of the Section. He was, however, informed by the Secretary of the Society that such a project was entirely outside the scope of the Section. The R.S.M.

under their charter were only allowed to deal with scientific subjects. However, several members of the Section met and during the year 1932 it was decided to form an independent body to be called the Association of Anæsthetists, having as its first objective the introduction of a diploma in anæsthetics. He has taken an active part in the work of the Association since its foundation and was one of the first examiners for the Diploma, the first examination for which took place in 1935. He is at the present time the senior member of the newly formed Faculty of Anæsthetists.

During the war Magill chose what was probably the hardest role of all, to remain in the bombed and war wracked metropolis. He narrowly escaped death, for the house in Wimpole Street in which he lived was destroyed by a bomb in 1941, but luckily at the time he was not in it. He carried on his attendances at both Westminster and the Brompton Hospitals throughout the war, and in addition he became adviser to the Ministry of Health, laying the foundation of the anæsthetic section of the present National Health Service.

Many well-merited honours have fallen to Magill. In 1945 his own University of Belfast conferred on him an Hon. D.Sc. In 1938 the Anæsthetic Section of the Royal Society of Medicine presented him with the Hickman Medal—the highest award which his brother anæsthetists could confer on him. In 1939 the Ulster Medical Society gave him a medal and elected him Robert Campbell Memorial orator. This year he was presented by the Irish Medical Graduates Association with the Arnott Commemoration Medal. His services to the Royal Family, which are many, were in 1946 recognized by the King, who was graciously pleased to invest him Commander of the Royal Victorian Order. In England

he is universally recognized not only as our most skilful exponent of the art, but as the doyen of the body of English Anæsthetists. Abroad, not only in the Dominions and America, but on the Continent of Europe, his name is well-known and held in highest esteem. British anæsthesia indeed owes its present position in no small measure to Ivan Magill.

DAMAGE TO THE SKIN DUE TO TRILENE

A search of the literature has not revealed an account of injury to the skin due to Trilene, therefore the following report may be of interest.

CASE REPORT

A fit woman aged 30 years underwent a vaginal repair operation in the lithotomy position. Anæsthesia was induced with 5 per cent thiopentone after the skin of the right antecubital fossa had been cleaned with a cotton wool swab dipped in spt. vini. meth. and an easy venipuncture performed. The syringe and swab were then placed upon the Boyles machine, which was used to maintain anæsthesia with semi-closed gas and oxygen. Later, using the same swab and the remains of the solution in the syringe, a further dose of thiopentone was given into the same vein to facilitate the suture of the levator muscles. Apart from when the injections were being given, the patient's arms were flexed upon the chest throughout the operation, which lasted 30 minutes.

On routine inspection of the patient in the ward 90 minutes later, it was noticed that an area of the right arm had sustained a first and second degree burn, and appropriate treatment was ordered. The illustration shows the condition of the arm on the second day after operation. Swelling of the arm occurred when infection supervened, but the healing was complete when the patient's gynaecological state permitted her discharge from the hospital on the 14th day after operation.

Inquiry showed that a swab soaked in Trilene and used for cleansing purposes elsewhere was placed on the anæsthetic trolley during the operation session. The anæsthetist also recollected that when preparing for the second injection on this particular patient there had been an excess of a blue fluid on the swab, but its importance was not apparent to him at the time.

By means of tests on members of the medical staff, it was

Trilene burn of arm—inadvertent cleansing of skin for vein puncture

shown that a burn resulted with Trilene and Trilene-spirit mixture. The latter was most severe and the degree varied with the time of application and the individual tested. The patient gave no history of any idiosyncrasy or allergic condition and as tests on other subjects were so definite it was not considered wise to subject her to an examination for susceptibility.

COMMENT

Whilst on active service a similar case was experienced when desquamation followed the use of Trilene as a cleansing agent prior to injection and its subsequent use was avoided. This repetition of injury lends emphasis to the need for care in the use of this fluid for such a purpose. It would be especially dangerous if, as is common practice, a wet swab was left to cover the point of injection.

Owing, no doubt, to the essential difference in the methods usually employed for their administration, this property of trichlorethylene is not so well recognized as it is in the case of the related substance trichlormethane (chloroform). A reaction between spirit and Trilene is considered unlikely, but chronic dermatitis in workers handling trichlorethylene has been experienced by manufacturing chemists.

I am indebted to Professor T. N. A. Jeffcoate for consenting to the publication of case.

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CORRESPONDENCE

The Editor, *British Journal of Anæsthesia.*
Sir,

In your January, 1950 issue you printed a note which I contributed on "Naso-Tracheal Tubes Held Up At The Cords: A Remedy".

May I point out that this remedy is successful only in adults and is of no value in children. In children I have repeatedly tried Professor Macintosh's method of flexing the head, also without success. No doubt hyperpnoea, induced with CO_2, would overcome the difficulty, but my patients are "curarized".

I would be glad to know if any of your readers, who have encountered this difficulty, have discovered any simple trick for overcoming it.

Yours faithfully,

J. G. BOURNE

St. Thomas's Hospital,
*London, S.E.*1.

REVIEWS

The Medical Annual 1949. Edited by SIR HENRY TIDY and A. RENDLE SHORT. 58 plates with 32 illustrations. John Wright & Sons Ltd., Bristol. 25*s*.

The Medical Annual is slightly smaller this year than last, contains less than half the number of illustrations, but a few more plates. It is as always a most useful book to have in one's possession; neither general practitioner nor specialist is likely to be able to understand all the articles but he can get some idea of what the various specialists are doing, each in his own line and to know to whom to apply for help when the occasion arises.

Dr. Langton Hewer writes the article on "Anæsthesia and Analgesia". He refers to the Oxemeter, an ingenious device for measuring the oxygen saturation of the arterial blood, but he does not mention the R.R.H. CO_2 indicator for the presence of CO_2, an account of which appeared in the *B. J. A.* in January, 1950.

It is interesting to see how ideas change as the years go by. Not very long ago curare was regarded as a deadly poison used only by cruel fiends to prevent tortured animals from resisting their manipulations, and the intravenous injection of procaine was frequently cited as the cause of a sudden and unexpected collapse. Now these two drugs are recognized as of great service to our suffering humanity, even if on occasion a patient relaxed with curare recovers from an insufficient dose of the accompanying anæsthetic.

The Physiological Basis of Oxygen Therapy. By J. H. Comroe, Jr., M.D., and Robert D. Dripps. Published by Charles C. Thomas, Springfield, Illinois, U.S.A., and by Blackwell Scientific Publications, Ltd., Oxford, England. Price 15*s.* 0*d.*

"How nicely got up" is the expression of one's first thought on seeing this small monograph. It is doubtful if any advance in anæsthetics in the last fifty years has been so important as the use of oxygen. To the student of anæsthesia then, a monograph on the physiological basis of oxygen therapy by a competent authority, and Professors Comroe and Dripps would come under that heading, is likely to be of considerable service. The stress they lay upon the difference between the tension of oxygen in the blood and its content is important. A fundamental fact of this nature might serve as the basis of a classification of anoxia, though perhaps not so simple as the one they have used. There is an extensive bibliography, but it seems odd to us that the name of J. S. Haldane does not figure in it.

TWENTY-SIXTH ANNUAL CONGRESS OF ANESTHETISTS

THE twenty-sixth Annual Congress of Anesthetists (sponsored by the International Anesthesia Research Society) will be held in London from September 3rd to 7th, 1951.

Visitors will register on the morning of September 3rd There is no registration fee. Short papers will be read on the other mornings and there will be demonstrations at various London hospitals. Scientific discussions and business meetings will be held in the afternoons. There will be an exhibition, by manufacturers, of apparatus and drugs. There are facilities for scientific exhibits by anæsthetists, for which no charge will be made.

Visitors will be entertained, in the evenings, by various academic bodies. The Congress will end, on September 7th, with a dinner, for which the charge will be £3 3*s*. 0*d*., inclusive of wines.

Attendance at the Congress is not limited to members of the Society. Those anæsthetists who wish to attend are requested to write, as soon as possible, to a member of the London Committee, stating whether they wish to read a paper or to exhibit, giving the provisional title of their paper and its probable length. Manufacturers should address their inquiries to Mr. Charles King.

Visitors from the United States and Canada should apply for accommodation to Mrs. Laurette McMechan. Other visitors should make their own arrangements through a travel agency or may apply to Dr. Ronald

Jarman. Early reservation is essential owing to the expected heavy booking by visitors to the London Exhibition. The party from the United States and Canada will make a motor coach tour of some of the important provincial centres of England and Scotland after the Congress, with a few days in Paris at the end of September. Other visitors wishing to join this tour should apply to Dr. Jarman.

London Committee:

Dr. R. W. Cope, University College Hospital, W.C.1.

Dr. Vernon Hall, King's College Hospital, S.E.5.

Dr. Ronald Jarman, Royal Cancer Hospital, S.W.3.

A. Charles King, Esq., 27 Devonshire Street, London, W.1.

Dr. C. B. Lewis, Gordon Hospital, S.W.1.

Dr. Geoffrey Organe, Westminster Hospital, S.W.1.

Dr. C. F. Scurr, Westminster Hospital, S.W.1.

Dr. W. D. Wylie, St. Thomas's Hospital, S.E.1.